AN INTERESTING CASE OF WOLFF-PARKINSON-WHITE SYNDROME

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ABSTRACT
Wolff-Parkinson-White syndrome is the most common of the preexcitation syndromes and is due to an accessory pathway connecting the atria and ventricles. It’s incidence in the general population is 0.1 to 0.3% and is associated with a sudden cardiac death risk of less than 0.6%. Atrial flutter or fibrillation is seen in 20-25% of WPW syndrome. Since the bypass tract does not have the same decremental conducting properties as the AV node, the ventricular response may be unusually rapid and may cause ventricular fibrillation. Acute management of WPW syndrome with atrial fibrillation with hypotension is DC cardioversion. Haemodynamically stable patients can be treated pharmacologically, however ativoventricular node blockers should be avoided in atrial fibrillation/flutter with WPW as they can favour conduction through the accessory pathway, potentially leading to ventricular arrhythmias.

Keywords: WPW Syndrome, AF with Wide Complex qrs, Pre Excitation

CASES
A 22-year-old male patient presented to the emergency department with sudden onset of palpitations and dizziness. There was no history of chest pain, dyspnoea, or syncope. He had no relevant medical history or family history of sudden cardiac death. He denied any use of regular medications, alcohol or illicit drugs. On physical examination, pulse rate was 150/min., irregularly irregular. His blood pressure was 90/ 60 mm Hg. No other abnormality was detected on physical examination. Cardiovascular examination was also normal. The electrocardiogram revealed following features (Figure 1).

Since the patient was haemodynamically unstable, he was managed with DC cardioversion of 200 Joules. The arrhythmia terminated, and subsequent 12-lead electrocardiogram revealed the following features suggestive of WPW type of pre-excitation syndrome (Figure 2):
1. Sinus rhythm with very short PR interval (< 120 ms)
2. Broad QRS complexes with a slurred upstroke to the QRS complexes — the delta wave.
3. A relatively normal, narrow ensuing terminal QRS deflexion.

Figure 1: Irregularly irregular wide qrs complex tachycardia.
Final Diagnosis
WPW syndrome with atrial fibrillation with fast ventricular rate. The patient was put on oral amiodarone 200 mg twice a day and discharged with the advice for further follow up for electrophysiological studies and radiofrequency ablation.

DISCUSSION
WPW syndrome is characterized by the presence of an accessory pathway, usually a bundle of Kent. The classic electrocardiographic presentation of WPW syndrome consists of:
1. A short PR interval.
2. A slurred, thickened initial upstroke of the QRS complex, which is termed as delta wave.
3. A relatively normal, narrow ensuing terminal QRS deflexion sometimes referred to as the main QRS deflexion.
4. Slight widening of the QRS deflexion.
5. Secondary ST segment and T wave changes

In 1930, Wolff, Parkinson and White reported 11 cases of young, healthy patients with normal hearts who presented with widened QRS, abnormally short P-R intervals, and paroxysms of tachycardia, including supraventricular tachycardia, paroxysmal atrial fibrillation and atrial flutter (Wolff et al., 1930). Atrial fibrillation in patients with WPW Syndrome is potentially lethal arrhythmia due to its potential to deteriorate into ventricular fibrillation (Vfib) (Klein et al., 1979).

In normal patients without an accessory pathway, the heart is protected from exceptionally high ventricular rates by the relatively long refractory period of the AV node. This generally limits the maximum ventricular rate. In patients with WPW, short anterograde refractory period of the accessory pathway, may allow faster transmission of impulses from the atrium and correspondingly higher ventricular rates can be reached. The rapid ventricular rate may not allow for adequate diastolic filling of the ventricle and this in turn can predispose to hypotension. In addition, sympathetic discharge secondary to hypotension can lead to an even shorter refractory period of the accessory pathway and subsequently increase the ventricular rate further. If the ventricular rate becomes too high, this can predispose to Vfib (Wolff et al., 1930). Afib usually does not conduct at a rate of more than 180 bpm through the normal AV node. On the other hand, conduction through an accessory pathway often results in more rapid ventricular rates. This usually appears as a bizarre, wide complex, irregular tachycardia on ECG with rates often in the 250 bpm range or higher as was noted in this patient. The impulses from the atria are conducted to the ventricles via either:
- Both the AV node and accessory pathway producing a broad fusion complex
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- Or just the AV node producing a narrow complex (without a delta wave)
- Or just the accessory pathway producing a very broad ‘pure’ delta wave.

Treatment of Atrial fibrillation associated with WPW is necessarily different than for a patient without an accessory pathway. In WPW associated Atrial fibrillation, the goal is to prolong the anterograde refractory period of the accessory pathway relative to the AV node. This slows the rate of conduction through the accessory pathway, thus the ventricular rate slows down.

In patients with non WPW associated Afib, the goal is to increase the refractory period of the AV node (slow conduction). Standard rate control by drugs that prolong the refractory period of the AV node (e.g., calcium channel blockers, beta-blockers, digoxin, and even adenosine) conversely result in a higher rate of transmission through the accessory pathway and paradoxically increase the ventricular rate. This could have dangerous consequences possibly causing the arrhythmia to deteriorate into Ventricular fibrillation. Thus, such drugs are contraindicated in WPW associated A fib.  

Antiarrhythmic Drugs: Amiodarone though recommended may accelerate ventricular rate or fibrillation and hence should be avoided as well (Sheinman and Evans, 1982; Boriani et al., 1996). Lidocaine administration generally has no significant effect or produces acceleration of ventricular response during AF, hence is unlikely to have beneficial effects and may be deleterious (Akhtar et al., 1981).

If the patient is hemodynamically stable, pharmacologic conversion with ibutilide, a class III antiarrhythmic drug available for intravenous administration (Glatter et al., 2001) or procainamide (Mandel et al., 1975) may be attempted. Ibutilide (1mg iv over 10 minutes) with monitoring of QT intervals for 6 hours post infusion. There is a 8% risk of torsades with ibutilide infusion. Procainamide at 30 mg/min with a maximum dose of 17 mg/kg iv infusion. Ibutilide is preferred because procainamide takes a long time to achieve therapeutic levels (about 60 minutes) and is associated with hypotension during rapid infusion.

If the patient is unstable, with evidence of hypoperfusion, primary synchronized cardioversion should be the first-line of treatment (this patient was successfully cardioverted).

In this case, the patient was deemed unstable and treated with synchronized cardioversion. His post cardioversion ECG shows the delta wave of WPW syndrome.

Conclusion

Pre-excited atrial fibrillation is a well recognized cause of sudden cardiac death, for which there is a potential “cure” in the form of radiofrequency ablation of the pathway. In patients presenting with hemodynamic instability secondary to atrial fibrillation with wwp, prompt synchronized cardioversion is the favored strategy. For hemodynamically stable patients pharmacotherapy with agents like ibutilide or procainamide may be utilized.

REFERENCES


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